A 60-year-old dentist presents to the emergency room with palpitations for 3 hours. The ECG shows atrial flutter (AFl) with atrioventricular (AV) conduction varying from 2:1 to 4:1. He has known hypertension for which he takes 10 mg of enalapril daily. In the last 2 years he was seen twice because of AFl and on both occasions 1 mg of ibutilide IV resulted in the return of sinus rhythm with 1:1 AV conduction. Catheter ablation of the arrhythmia is discussed with the patient, to which he agrees and curative AFl ablation is scheduled in 4 weeks.

**History**

In 1906, Einthoven made an electrocardiographic recording of AFl. In 1913, Lewis called attention to the typical saw tooth pattern and the negative deflections of the atrial waves in leads II and III.

During the next 50 years, there was much discussion about whether AFl was caused by a rapidly firing atrial focus or is the result of a large circus movement involving the atria. After mapping atrial activation with endocardial and esophageal recordings, Puech et al concluded that the flutter cycle in the human heart involved activation of the whole right atrium. Stimulation studies by Waldo et al revealed that in postoperative AFl the arrhythmia was based on a reentry mechanism involving a large atrial area. They demonstrated the presence of an excitable gap that allowed the speeding up of the flutter rate during atrial pacing and termination of the arrhythmia by pacing.

**Mechanism and Types of Macroreentrant Tachycardias**

With the use of endocardial activation mapping and stimulation studies, several investigators have shown that different types of macroreentrant atrial tachycardias are possible. As indicated in Figure 1, the circuit is usually located in the right atrium. In the circuit, a critical component of slow conduction is frequently present, often located in the isthmus of right atrial tissue, which is between the tricuspid annulus, the inferior vena cava, and the coronary sinus with the eustachian valve and ridge. Occasionally, the circuit curves around a scar or surgical incision in the atrium or is confined to the left atrium. Endocardial activation mapping and stimulation studies are required to determine the circuit. Of the different types of AFl shown in Figure 1, the typical, common, or type 1 flutter is the most frequent. In this type of flutter, right atrial activation rotates in a counterclockwise direction. Less common are reverse (clockwise) typical flutter, lower loop flutter, and macroreentrant tachycardias around a scar in the atrium or a circuit in the left atrium. The first 3 types incorporate the right atrial isthmus into the circuit and one might restrict the label AFl to these 3 because of the implications for pharmacological and ablative therapy.

**Profile of the Patient and Electrocardiographic Findings**

Although the exact incidence of AFl is not known, it is a common arrhythmia estimated to be present in approximately 10% of patients presenting with a supraventricular tachycardia. AFl seems to occur more often in men than in women. Additional abnormalities are frequently present, such as hypertension, coronary artery disease, valvular heart disease, the sick sinus syndrome, previous cardiac surgery, and chronic lung disease. The frequent association between AFl and other cardiac defects necessitates an echocardiographic examination. The acute occurrence of AFl can impair cardiac function, lower blood pressure, and initiate myocardial ischemia. Permanent AFl with a rapid ventricular rate may lead to a tachycardia-mediated cardiomyopathy. Tolerance of the rapid ventricular rate during AFl will be influenced by the presence of additional cardiac or pulmonary abnormalities.

Electrocardiographically, AFl has a nearly constant rate of 250 to 350 beats per minute. In the absence of drugs that lengthen the refractory period of the AV node, there is a 2:1 relation between atrial and ventricular activity. In typical AFl, global atrial activation, as seen in the extremity leads,
occupies the entire interval between two atrial depolarizations. In the precordial leads, which record more local depolarization, shorter atrial complexes may occur. Figure 2 shows examples of common or typical (counterclockwise) and reverse typical (clockwise) AFI. Both are right atrial isthmus dependent. Typical AFI usually has negative flutter waves in leads II and III and positive atrial deflections in lead V1. In reverse AFI, positive flutter waves are present in the inferior leads and negative atrial deflection in lead V1. The other types of atrial macroreentrant tachycardias have less characteristic ECG patterns.

Management of the Arrhythmia
The effect of AFI on the hemodynamic condition of the patient determines whether immediate conversion to sinus rhythm should be attempted (by electrical cardioversion or atrial overdrive pacing) by pharmacological treatment.

Pharmacological Therapy
Perpetuation of AFI requires that the tachycardia circuit be longer than the product of conduction velocity of the circulating impulse and the duration of the refractory period in the circuit (the so-called wave length). To terminate the arrhythmia, therefore, a drug should be given which does not slow conduction velocity but rather prolongs the refractory period within the tachycardia circuit, inhibiting continuation of the circulating wave front.9–11

Class III drugs, such as ibutilide, dofetilide, azimilide, and sotalol are typically used. For acute termination of AFI, intravenous ibutilide is most effective.12,13 For prevention of recurrence, 2 aspects have to be considered. The arrhythmia is usually caused by one or more atrial premature beats that can be prevented by class I drugs (flecainide, procainamide, quinidine), class III drugs, and amiodarone. Maintenance of the arrhythmia can be prevented by prolonging the wave...
length of the circulating impulse by class III drugs. For ventricular rate control during AFl, drugs should be prescribed that prolong the refractory period of the AV node, such as β-blocking agents, calcium antagonists, digitalis, and amiodarone (Table).

When deciding on pharmacological therapy, side effects and the possible dangers of drug administration should be considered. Class III drugs can produce torsade de pointes arrhythmias. This occurs in 1% to 4% of patients and is related to dose and presence of abnormalities in cardiac and kidney function. Class I drugs may lower the flutter rate, resulting in 1:1 AV conduction and a much faster ventricular rate. Especially in the case of class I-C drugs, this may be accompanied by marked QRS widening and initiation of ventricular tachycardia. QRS widening on ventricular rate increase may vary individually. It is therefore advisable to perform an exercise test after treatment with a class I-C drug has been initiated. If QRS widening on ventricular rate increase occurs, the class I-C drug should be discontinued, and/or a drug prolonging the refractory period of the AV node should be administered.

Nonpharmacological Therapy

Electrical Cardioversion and Overdrive Pacing

External electrical cardioversion has been used to terminate AFI since its introduction by Lown et al. The method is safe and effective, terminating AFI in >90% of episodes. Shock strength during cardioversion of AFI is usually started at 50 joules; a direct current, preferably biphasic shock, is preferred. Because external electrical cardioversion requires anesthesia, some doctors and patients prefer atrial overdrive pacing to terminate AFI. This also has the advantage of being able to pace in the case of sick sinus syndrome after termination of AFI. Implantable devices have become available, allowing both antitachycardia and 50-Hz burst pacing, as well as (when needed) internal cardioversion of AFI.

Catheter Ablation

The identification of the macroreentrant nature of AFI and the ability to localize the circuit by endocardial activation mapping and pacing resulted in attempts to interrupt the circuit by ablative interventions. Initially, high-energy shocks were given endocardially, followed by local administration of radiofrequency current. Nowadays, catheter ablation of AFI has become a safe, curative, and highly successful procedure, particularly when the right atrial isthmus is incorporated in the flutter circuit. Demonstration of bidirectional isthmus block after ablation predicts a high long-term success. Catheter ablative therapy results in either no or very minimal enzyme rise, and complications are rare. Occasionally, ST-segment elevation in the inferior leads can be observed during catheter ablation but without right coronary angiography.

Atrial Flutter and Atrial Fibrillation: Frequently Associated, but Not the Same Arrhythmia

Episodes of AFI and AF often occur in the same patient, but as stressed by Cosio and Delpon, these are different arrhythmias both mechanistically and therapeutically. AFI is a macroreentrant arrhythmia; AF is either focally determined or based on multiple reentrant wavelets. Different drugs are required for termination of each arrhythmia: class III drugs for AFI and class I for AF. Likewise, different nonpharmacological approaches are required for control of the arrhythmia. Occasionally, AF occurs when AFI (the so-called mother wave) deteriorates into multiple small reentrant wavelets. In that situation (Figure 3), treatment with a class I-C drug (amiodarone) may terminate AF but reinitiate AFI. In this subgroup, right atrial isthmus ablation with continuation of the same drug regimen often results in control of both AF and

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<table>
<thead>
<tr>
<th>Pharmacological Therapy of Atrial Flutter</th>
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<tbody>
<tr>
<td><strong>Termination</strong></td>
</tr>
<tr>
<td>Ibutilide: &gt;60 kg: 1 mg IV in 10 min;</td>
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<tr>
<td>&lt;60 kg: 0.01 mg/kg IV in 10 min</td>
</tr>
<tr>
<td>Sotalol: 1 mg/kg IV in 10 min</td>
</tr>
<tr>
<td>Amiodarone: 5 mg/kg IV in 10 min</td>
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<tr>
<td><strong>Prevention of recurrences</strong></td>
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<tr>
<td>Preventing the initiating atrial</td>
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<tr>
<td>premature beat(s)</td>
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<tr>
<td>Class I-A and class I-C drugs</td>
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<tr>
<td>β-Blocking agents</td>
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<tr>
<td>Amiodarone</td>
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<tr>
<td><strong>Prolonging the atrial refractory</strong></td>
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<tr>
<td>period</td>
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<tr>
<td>Class III drugs</td>
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<tr>
<td><strong>Ventricular rate control</strong></td>
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<td>β-Blocking agents</td>
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<td>Calcium antagonists</td>
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<tr>
<td>Digitalis</td>
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<td>Amiodarone</td>
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Figure 3. AF (A) changes into AFI after the administration of the class I-C drug flecainide (B).
In patients with episodes of both AFI and AF, flutter ablation may have different effects on the incidence of AF.\(^2\)\(^7\)-\(^2\)\(^9\) Nabar et al\(^{30}\) noted a marked reduction in AF episodes after flutter ablation in patients with AFI as their dominant arrhythmia. This was not observed when AF occurred more often than AFI.

**Anticoagulant Therapy?**

Except in a few selected patients, anticoagulation should be prescribed in the patient with paroxysmal or permanent AF. For example, the patient with lone AF and normal LV function can probably be treated with aspirin alone.

What about AFI? Unfortunately, no prospective randomized studies are available to tell us about the incidence of thromboembolic complications in paroxysmal and permanent AFI and the value of anticoagulant therapy. There are reasons, however, to consider anticoagulation for the patient with AFI. First, these patients frequently have episodic AFI. Second, associated abnormalities are often present that favor thromboembolic complications such as valvular disease, hypertension, and heart failure. Third, as shown by Jordaens et al,\(^{31}\) delayed restoration of atrial function occurs after conversion of AFI by pacing or electrical shock. It is therefore advisable to anticoagulate the AFI patient unless the duration of the arrhythmia is <48 hours or lone AFI with preserved left ventricular function is present, in which case aspirin could be given, as noted above. After successful catheter ablation of AFI, anticoagulant therapy can be stopped 4 to 6 weeks later if sinus rhythm is still present and there are no other indications for its continuation.

**Conclusion**

Natale et al\(^{32}\) have suggested catheter ablation as the first-line therapy in patients with AFI and normal or mildly enlarged left atrial size. They found a higher success rate, better quality of life, lower occurrence of AF, and less need for rehospitalization at follow-up when compared with drug treatment. Such an approach requires an experienced electrophysiologist and comprehensive catheterization facilities. In practice, therefore, pharmacological therapy and electrical cardioversion will remain the most used initial treatment for AFI. It is likely, however, that the increasing availability of catheter ablation will lead to greater use of this curative intervention.

**References**


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